ELECTROPHYSIOLOGIC FINDINGS AFTER STAGED PALLIATION FOR HYPOPLASTIC LEFT HEART SYNDROME

Cheryl C. Kürer, M.D., Victoria L. Vetter, M.D, F.A.C.C., Children's Hospital of Philadelphia, Philadelphia, PA.

Eight pts with hypoplastic left heart syndrome status post Norwood palliation and modified Fontan repair were studied to determine the electrophysiologic consequences of these procedures. Electrophysiologic studies were performed 12-74 months (median 13.5) following Fontan repair at a median age of 2.4 years. Resting bradycardia was present in 3 pts; 2 had junctional or ectopic atrial escape rhythms and 1 had sinus bradycardia. The corrected sinus node recovery time and sinoatrial conduction times were normal in all 6 pts in sinus rhythm. The 2 pts not in sinus rhythm had prolonged corrected pacemaker recovery times at 440 and 1920 ms. Effective atrial refractory periods (ARP) were normal in all pts in their basal rhythm and at paced cycle lengths (PCL) of 600 and 500 ms. At a PCL of 400 ms, 3 pts had prolonged effective ARP. Functional ARP were abnormal in 1 pt at a PCL of 600 ms and in 2 pts at a PCL of 400 ms. Atrial catheter endocardial mapping in 7 pts confirmed the site of pacemaker origin, and demonstrated delayed activation of adjacent atrial sites in 2 of the 6 pts in an atrial rhythm. Programmed atrial stimulation induced sustained intraatrial reentry (CL 410 ms) in 1 pt and nonsustained atrial flutter (CL 220 ms) in 1; both pts also had sinus node dysfunction and prolonged atrial refractoriness. Atrioventricular nodal function was normal in all pts. Ventricular stimulation was performed in 2 pts and did not induce arrhythmia.

We conclude that abnormalities of sinus node function, atrial conduction and atrial refractoriness are present following staged palliation for hypoplastic left heart syndrome. These abnormalities provide the electrophysiologic substrate for the development of sick sinus syndrome and supraventricular tachyarrhythmias.

Al+++ BINDING STUDIES AND PREVENTION OF BIOPROSTHETIC HEART VALVE CALCIFICATION

Catherine L. Webb, M.D., William E. Flowers, B.S., James Boyd, B.A., Allen C. Alfrey, M.D., Frederick J. Schoen, M.D., Robert J. Levy, M.D. University of Michigan Medical Center, Ann Arbor, MI

Calcification (CALC) frequently causes failure of glutaraldehyde pretreated bioprosthetic heart valves (GPBP). Al+++ has been shown to inhibit GPBP CALC in the rat subdermal model. This study was designed to determine the GPBP tissue Al+++ level necessary to effect long term CALC inhibition. Specimens of glutaraldehyde pretreated bovine pericardium were incubated (1hr, 25C) in 0.01 $AlCl_3$, or 0.05M HEPES buffer, then subdermally implanted in weanling male rats (50-60gm) for 21 and 60 days. Preimplant GPBP Ca++ and Al+++ levels were 0.58±0.02ug/mg and 2646±178ug/gm respectively. GPBP Al+++ levels showed a progressive decline after 21 and 60 day explants (743±80ug/gm, 363±49ug/gm respectively). Calcification was inhibited at each time point (Ca++=2.33±0.17ug/mg, 11.57±4.58ug/mg, respectively) compared to control. No adverse effects were noted. Electron energy loss spectroscopy showed Al+++ to be localized intracellularly at phosphorus rich sites. We conclude that GPBP Al+++ levels ≥ 363 ug/gm are necessary for calcification inhibition.

MYOCARDIAL INTERSTITIAL AND CORONARY VENOUS EFFLUENT ADENOSINE LEVELS DURING SEVERE HYPOXIA IN THE DEVELOPING RABBIT.

G. Paul Matherne, M.D., Sharon D. Coleman, B.S., John P. Headrick, Ph.D., Robert M. Berne, M.D. Univ. of Virginia Medical School, Charlottesville, Va.

Adenosine (ADO) has a role in coronary blood flow control in both the mature and immature heart. Study of this role depends on accurate estimates of ADO in the interstitial space. To determine developmental differences and changes with hypoxia, we measured ADO in the myocardial interstitial fluid (ISF) and coronary venous effluent (CVE) in isolated hearts from 10 immature (IM) (age 2-4 wks) and 11 mature (M) rabbits during normoxia (N) and severe hypoxia (H) at a constant coronary flow. Estimates of ISF-ADO (nanomolar) were obtained with epicardial porous discs.

Results:

	ISF-ADO		CVE-ADO	
	N	H	N	H
IM	129±16**	1180±231*	30±9**	1389±207*,†
M	228±35**	1225±300*	61±13**	836±102*

*P<.01, **N ISF-ADO<H CVE-ADO, †IM CVE-ADO<M CVE-ADO

No age related differences were present in ISF-ADO during normoxia or hypoxia even though the immature hearts had significantly lower coronary resistance, lower contractility and performed less work than mature hearts. All ADO levels increased during hypoxia and were associated with a decrease in coronary resistance. The ADO gradient from ISF to CVE during normoxia was abolished during hypoxia in both age groups. During hypoxia CVE-ADO was greater in immature than mature hearts.

Conclusion: 1) Despite developmental differences in resistance, function and work, ISF-ADO is similar in immature and mature rabbits.

2) The ISF to CVE gradient for ADO is abolished during hypoxia in both ages but in the immature rabbit this leads to higher CVE-ADO levels than in the mature rabbit.

Wednesday, March 21, 1990
Poster Displayed: 9:00AM–12:00NOON
Author Present: 11:00AM–12:00NOON
Hall C, New Orleans Convention Center
Cardiac Surgery: Coronary and Valve Disease

LASER REVASCULARIZATION OF THE ISCHEMIC MYOCARDIUM

M. Mirhoseini, M.D. S. Shelgikar, M.D. Mary M. Cayton, R.N. Heart & Lung Institute of Wisconsin, Milwaukee, WI

The concept of revascularization of the myocardium by transventricular CO_2 laser channels was studied in depth in the experimental laboratory and then brought to the clinical level. Patients who are candidates for this procedure are those with diffuse coronary artery disease, those who have had poor results from previous surgery, or those who do not respond to other forms of treatment. CO_2 laser channels were made in viable but ischemic areas of the LV, the coronary artery perfusing the area was not amenable to bypass graft or angioplasty techniques. Perfusion occurs from the LV through the system of myocardial sinusoids. To date this procedure has been performed on 16 patients. Followup time ranges from a few months to 4½ years. Evaluation includes pre and postoperative cineangiograms, thallium stress test, perfusion patterns by radionucleide techniques, and analysis of ventricular function. Preliminary results indicate the channels remain patent. Improved LV function and increased uptake of radionucleides can be demonstrated. There was no operative morbidity or mortality, there has been one late death (4 years) due to carcinoma of the colon. Early clinical experience indicates this may be a viable method of treating patients with ischemic heart disease who are not candidates for other forms of medical or surgical management.